Method andmaterials: We were observed 316 (male) student, who arrived from all province of Mongolia in Ulaanbaatar. They are divided into 4 groups by date of birth: 1. Born before 1987; 2. Born in 1988–89; 3. Born in 1990; 4. Born after 1991 (after launching HBV vaccination), In all serum samples tested for HBsAg, anti-HBc and anti-HBs by ELISA.
Results: The results of HBV markers test are presented in the table.

Group	n	HBsAg %	Anti-HBc %	Anti-HBs %
1	44	6.8	34.1	34.1
2	111	5.4	27.9	27.0
3	71	7.0	21.1	22.5
4	91	3.3	6.6	14.3

The data in the table show that, the exposure ofHBV infection in non vaccinated more than 20%, then vaccinated populations less than 7%.
Conclusion: The exposure and prevalence rate of HBV infection significant difference in vaccinated and non vaccinated population.

PP-120 Cloning and screening transregulated genes of HBEBP2 by SSH

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Objective: To screen and identify human genes transactivated by HBEBP2 by constructing a cDNA subtractive library with suppression subtractive hybridization (SSH) technique.
Methods: pcDNA3.1(–)-myc-his-HBEBP2 was constructed. Then the L02 cells was transfected by pcDNA3.1(–)-myc-his-HBEBP2 and pcDNA3.1(–)-myc-his empty vector, and SSH method was employed to analyze the differentially expressed DNA sequence between the two groups. After restriction enzyme *Rsa* I digestion, small sizes cDNAs were obtained. Then tester cDNA was divided into two groups and ligated to the specific adaptor 1 and adaptor 2, respectively. Aftertester cDNA was hybridized with driver cDNA twice and underwent two times of nested PCR, the second PCR production was subcloned into pGEM-Teasy plasmid vectors to set up the subtractive library. Amplification of the library was transformed into *E. coli*. The cDNA was sequenced and analyzed in GenBank with Blastn search after PCR.
Results: The subtractive library of genes transactivated by HBEBP2 was constructed successfully. The amplified library contains 80 positive clones containing 200 bp-1000 bp inserts. Sequence analysis was performed in 26 clones randomly, and the full length sequences were obtained with bioinformatics method. Altogether 7coding sequences were gotten.
Conclusions: The obtained sequences may be target genes transactivated by HBEBP2, and some genes coding proteins involved in metabolism, autoimmunity regulation, protein modification, cell cycle regulation.

PP-121 Construction and expression of a novel HBeAg binding protein 1 of hepatitis B virus in yeast

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Objective: To construct the eukaryotic expression vector of HBEBP1 gene and express HBEBP1 recombinant protein in yeast.
Methods: PCR was performed to amplify the gene of HBEBP1 from the cDNA template origining from HepG2, and the gene was cloned into pGEM-T vector. After sequencing, the correct DNA fragment was cut from pGEM-T-HBEBP1 and inserted into yeast expression plasmid pGBKT7. The reconstructed plasmid pGBKT7-HBEBP1 was transformed into yeast cell AH109 and screened on the synthetic dropout nutrient medium (SD/-Trp/Kana). The yeast protein was isolated and analyzed with SDS-PAGE and Western blot.
Results: The eukaryotic expressive vector was constructed successfully. The results of Western blot showed HBEBP1 protein was existed within yeast cells and the molecular weight of it was about 32.3kD.
Conclusions: The successful expression of HBEBP1 protein in yeast cells lay the foundation for studying biological function of HBEBP1.

PP-122 Association between IRF-3 polymorphisms and susceptibility to chronic hepatitis B virus infection

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Aim: To investigate the association between the three tagSNPs (rs10415576, rs2304204, rs2304206) of IRF3 gene and genetic susceptibility of chronic hepatitis B virus (HBV) infection in Chinese patients.
Materials and Methods: Based on the case-control study in 985 case of chronic HBV infection and 294 self-limiting HBV infection individuals as controls, three tagSNPs in the IRF3 gene were genotyped by Multiplex Snapshot technique. The genotype and allele frequencies were calculated and analyzed.
Results: The three SNPs sites showed no significant genotyping/allelic associations with chronic HBV infection. Overall allelic P values: rs10415576, $P = 0.0908$, OR (95%CI) = 1.1798 (0.9740–1.4291); rs2304204, $P = 0.5959$, OR (95%CI) = 1.0597 (0.8552–1.3133); rs2304206, $P = 0.8372$, OR (95%CI) = 1.0250 (0.8097–1.2976). Overall genotype P values: rs10415576, $P = 0.2106$; rs2304204, $P = 0.8458$; rs2304206, $P = 0.8315$. There was no statistically significant difference between chronic HBV infection and controls. Haplotypes generated by these three SNPs did not show significantly difference between the two groups either ($P > 0.05$).
Conclusion: The data suggest that the three tagSNPs sites of IRF3 gene are not associated with HBV infection in Chinese Han population.

PP-123 The role of HBeAg in the expression of B7-H1 on monocytes and IFN-γ in T cells

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Objective: To study the relationship between HBeAg and persistence of HBV infection.
Methods: Taking HBeAg negative CHB patients as the research objects, determined the expression of B7-H1/PD-1 and TLR-2 on $CD14^+$ PBMCs and IFN-γ and IL-4 in $CD3^+CD4^+$ cells before and after incubation with HBeAg and TLR-2

on CD14$^+$ PBMCs under the stimulation of HBeAg with or without anti-HBe antibody by flow cytometry.
Results: Both in CHB patients and healthy people, TLR-2 decreased significantly (t scores were 4.948 and 3.245, both P values were below 0.05) and B7-H1 increased (t scores were 3.612 and 2.341, both P values were below 0.05) and IFN-γ decreased significantly (CHB group, $t = 2.184$ and $P = 0.050$, healthy control group, $t = 2.800$ and $P = 0.030$) after incubation with HBeAg. TLR-2 also decreased under the stimulation of HBeAg with anti-HBe antibody, but there was no statistical difference compared with incubation with HBeAg alone ($P > 0.05$).
Conclusions HBeAg could increase the negative costimulatory molecule B7-H1, inhibit TLR-2 and IFN-γ, these lead to the reduced capacity of specific CTLs in viral clearance.

PP-124 The variation of HBsAg titre in patients with chronic hepatitis B, HBV-related liver cirrhosis and hepatocellular carcinoma

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Objective: To analyze HBsAg titre variational tendency in patients with chronic hepatitis B (CHB), HBV-related liver cirrhosis (LC) and hepatocellular carcinoma (HCC), and the correlation with HBV DNA in each disease.
Methods: HBsAg titre and HBV DNA were measured in 47 CHB, 72 LC and 54 HCC patients used Abbott chemiluminescence and a real-time PCR respectively.
Results: HBsAg titre level in serum decreased from CHB [2361.10 (975.69–5427.96) IU/mL] to LC [1001.64 (318.72–1819.99) IU/mL] and HCC [594.35 (133.75–1337.00) IU/mL], and there were statistically significant different ($\chi^2 = 24.394$, $P < 0.001$). Moreover, HBsAg titre was significantly higher in CHB than LC ($Z = -3.754$, $P < 0.001$), CHB had significantly higher HBsAg titre than HCC ($Z = -4.630$, $P < 0.001$), however, LC and HCC had no statistically significant different ($Z = -1.720$, $P = 0.085$). In HBeAg positive patients, HBsAg titre decreased from CHB [3259.83 (1710.60–7547.85) IU/mL] to LC [1077.30 (374.39–2553.59) IU/mL] and HCC [789.72 (162.97–1470.95) IU/mL], and there were significant different ($\chi^2 = 15.643$, $P < 0.0001$). In HBeAg negative patients, the decline of HBsAg titre among CHB [1669.00 (240.78–2920.97) IU/mL] to LC [1001.64 (115.91–1609.59) IU/mL] and HCC [582.05 (133.75–1209.07) IU/mL] had significant different ($\chi^2 = 6.423$, $P = 0.04$). Correlation between HBsAg titre and HBV DNA was found in CHB ($r = 0.297$, $P = 0.043$), LC ($r = 0.346$, $P = 0.003$) and HCC ($r = 0.452$, $P = 0.001$), respectivly.
Conclusion: HBsAg titre level in serum decreased progressively from CHB to LC and HCC group. There were positive correlations between HBsAg titre and HBV DNA level in CHB, LC and HCC.

PP-125 Chinese herbal medicine long-term anti-HBV infection personalized treatments

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Objectives: Investigate the efficacy of a Chinese herbal compound prescription (CHCP) for 32 HBeAg positive chronic hepatitis patients' two year anti-HBV infection treatments.
Methods: The main ingredients of the CHCP have Chaihu, Huangqi and Diding. The main prescription consists of 11 herbal ingredients, which was used for all patients. The sub-prescription consists of 2–4 herbal ingredients, which was used for patients based on their different symptoms. The patients took the decocted CHCP two times each day at least half one hour after meals.
Results: Of 32 CHCP-treated patients, ten (31.2%) have obtained HBeAg seroconversion, ALT normalization, and with HBV DNA <1000copies/mL (lower limit of detection). These patients' baseline average characters were Age = 25.8 years old, ALT = 197.74U/L, HBV DNA = 5.71e7 copies/ml, and HBeAg = 551.25S/CO. The average time to achieve HBV DNA <1000 copies/ml was 72 weeks. The other 22 CHCP-treated patients' baseline average characters were Age = 35.5 years old, ALT = 148.75U/L, HBV DNA = 4.26e7copies/ml, and HBeAg = 796.99S/CO. For all CHCP-treated patients, neither demonstrated substitutions associated with NTCB resistance or the common adverse events and renal events.
Conclusion: Two years CHCP anti-HBV Infection therapy is quite effective for the HBeAg positive younger patients whose baseline ALT >200U/L, HBeAg <500S/CO, and HBV DNA <1e8copies/ml. The CHCP is safe for long-term's anti-HBV infection personalized therapy.
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PP-126 Regulation of TLRs, PD-1/PD-L1 expression and cytokine secretion on peripheral monocytes by hepatitis B virus E antigen

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Background: HBeAg is important for establishment of persistent infection. The aim of this study was to detect the regulation of TLRs, PD-1/PD-L1 expression and cytokine secretion on PBMCs by HBeAg.
Methods: Anti-HBe serum was collected from CHB with anti-HBe positive; PBMCs were isolated from healthy persons; Cytokines secretion were detected by CBA; RT-PCR examined TLR2, TLR4mRNA.
Result: CD14$^+$ PBMCs of TLR2 was significantly reduced in HBeAg-group compared to control ($p = 0.003$). TLR4 expression on CD3$^+$ cells in HBeAg-group was significantly decreased, but expressions of PD-1 and PD-L1 were significantly up-regulation; However, when HBeAg was reactivity against by Anti-HBe, the expression of TLR4, PD-1, PD-L1 on CD14$^+$ cells and CD3$^+$ cells were improved. At the same time, cytokines expression were detected, HBeAg was able to significantly down-regulate IL-2, IL-17 and IFN-γ, but up-regulate IL-10 ($P < 0.005$). RT-PCR illustrated that HBeAg was able to significantly down-regulate TLR2 mRNA on PBMCs ($p = 0.001$), while HBeAg was blocked with anti-HBe, the expression TLR2 mRNA was improved ($p < 0.05$).
Conclusion: HBeAg was able to significantly suppress TLR2 and TLR4 expression and mRNA on CD14+ PBMCs and PD-L1/PD-1 expressions on PBMCs, yet HBeAg blocked with anti-HBe could improve the expression of mRNA and decrease the expression of PD-L1 and PD-1; HBeAg was able to significantly down-regulate proinflammatory cytokines. These results promoted that HBeAg may be through the way suppressed expression of inflammatory factors genes induced by TLRs signal pathway and regulated secretion of cytokines to establish persistent infection in chronic hepatitis B patients.